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rest of a few months often brings such relief that further treatment is abandoned; because a remission which may naturally come in the course of the malady is too apt to satisfy the patient or the friends that the disease is at an end; and because our therapeutic measures have thus far proved, as a rule, so ineffective that we cannot often get our patients to consent to a great sacrifice of time and money, and all that goes with it, for a cure that at best may mean only comfortable uselessness for many years. The travelling or stimulating life usually suggested ends in aggravation of symptoms. Entire mental rest in a quiet place in a sedative climate, with simple food, abundance of sleep and moderate exercise, results in such improvement that there is every reason to suppose that such measures, if fully carried out, might do more."

PATHOLOGY.

Die pathologische Anatomie der Dementia paralytica. E. MENDEL. Neurologisches Centralblatt, 1890, No. 17, p. 519.

In the section for Neurology and Psychiatry of the Tenth International Congress Prof. Mendel gave a review of the present condition of the pathology of *Dementia paralytica*. The extended and careful researches of the last ten years lead him to think that further light on this subject cannot be expected, at least from the methods of investigation at present at command. He passes over without consideration all the changes in the skull, dura and pia about which there is no dispute, as well as the gross anatomical relations, such as atrophy of the convolutions and diminution in brain weight, and considers only the results of the microscopical examination of the brain.

The Neuroglia. Increase of the nuclei is a very common occurrence. The spider-cells also frequently show a great increase and extension. In this connection Golgi's staining has given especially beautiful results. Nuclei as well as spider-cells are present in normal brains, but in smaller numbers, and the last only of very small dimensions, while in general paralysis they exceed the normal size three or four times, or even more. In the normal brain spider-cells are mostly clearly seen only under the surface of the brain, while in general paralysis they are scattered throughout the entire thickness of the cortex. This last condition is also sometimes found in the neighborhood of encephalitic deposits and syphilitic neoplasms, but in these cases only in circumscribed places, while in paralysis it is more extended in the frontal and parietal lobes, especially in the central convolutions, and also in the basal portions of the frontal lobes. It is this spider-cell development which, pushing its way through the white substance, finally comes out on the exposure of ventricles as ependyma proliferation. The brain substance in long continued cases finally falls away in a confusion of fibres: sclerosis; if this process is strongly developed in the medullary laminæ, especially if the autopsy is made somewhat long after death, and the cortex has undergone a slight post mortem softening, the cortex can be separated from the medullary substance with the back of the scalpel, as Baillarger, Rey and Tuczek have pointed out. The separation as a rule takes place in the cortex itself, so that pieces of this remain attached to the medullary substance.

The Vessels. The larger brain vessels in general paralysis are more frequently intact, or show only a trifling amount of change, at times being more or less atheromatous. With the present methods of investigation it is in many cases difficult to say anything accurate and trustworthy of the condition of the small arteries and capillaries which lie in the ground substance. Notwithstanding this, however, it has been possible hitherto in most cases of paralysis to point out certain morbid changes in the vessels: increase of the nuclei in the vessel walls and

perivascular spaces, which Weidl observed as far back as 1859, miliary aneurisms, widened spindle-form adventitial spaces, etc. Here belongs also, as a widening of the lymph spaces, the cystoid degeneration so often found in general paralysis. Further changes in the vessel walls are the colloid and hyaloid degenerations described by Grieff and lately by Dagonet. All these changes occur also in other forms of brain disease, they have nothing characteristic of general paralysis, but it seems clear that with better means of investigation their *regular occurrence* in general paralysis can be shown.

The Ganglion Cells. At the session of the society of German Alienists in Berlin in 1883, Mendel took the ground, against strong opposition, that changes in the ganglion cells were an almost regular occurrence in general paralysis, and now the views of authors are almost unanimous that changes in the ganglion cells may always, or at least almost always be observed. Nissl's staining has materially advanced knowledge in this direction. A description of these changes was given by Mendel in 1884 (*Neurolog. Centralbl.*, 1884, p. 487). Widening of the pericellular spaces, changes in the protoplasm such as fatty pigment degeneration, sclerosis and atrophy of the cells (hyaline degeneration of Liebmann), changes in the nuclei such as shrinking or enlargement, are the usually reported findings. These changes are especially marked in the frontal lobes, on their lateral and medial surfaces, as well as on their lower; also in the parietal lobes, and least in the occipital lobes. Normal cells often lie between the altered ones. Similarly altered cells are also observed in other pathological processes of the brain.

The Nerve Fibres. To Tuczek belongs the credit of first calling attention in 1883 to the atrophy of medullated nerve-fibres in the cortex of paralytics. A large series of observations has been made in this direction with the following results: 1. The shrinking of the nerve-fibres in general paralysis is not confined to the cortex alone, but takes place throughout the entire brain. The nerve atrophy in the cortex is thus only one part of a process affecting the entire medulla of the brain and cerebellum. 2. This fibre-atrophy, especially the cortical, occurs not simply in progressive paralysis, but also in epilepsy, senile dementia, alcoholic paranoia, etc. Finally, reference may be made to the alteration of the nuclei of the brain nerves and to the degeneration of the peripheral nerves. With regard to the cord, this is normal in a number of cases of general paralysis, but in the great majority of cases it also is diseased. The changes that show themselves are: 1. Isolated gray degeneration of the posterior columns. 2. Isolated degeneration of the pyramidal tracts. 3. Disease of the pyramidal and cerebellar tracts with intact posterior columns. 4. Various combinations of diseases in different tracts.

From the anatomical findings described it is evident that no individual one of these morbid changes, either in the neuroglia, in the vessels, in the ganglion cells, or in the nerve fibres, is in and by itself characteristic of general paralysis; its essential difference from all other brain diseases lies not in the special changes, but in the diffuseness of the process, which appears spread over a great part of the cortex—sometimes over its whole extent, and even over the cerebellum, and taking all these facts into consideration we are justified in looking on general paralysis as a disease *sui generis*. In this it is self-evident that the clinical picture must vary according as the development is acute or chronic, according to the greater or less extension and according to the complications with other affections. The more closely and carefully the examination is made, the more are the frequent early negative reports of the microscopic examination invalidated, and now it can only rarely be asserted that in a well-marked case of general paralysis that has lasted a long

time there are not the characteristic findings in the neuroglia, the vessels, the ganglion cells, and the nerve fibres.

Up to this point Mendel's views are in essential accord with those of most alienists, but there is a dispute as soon as the question arises as to where the starting point of the morbid changes is to be sought. Regarding this there are two views directly opposed to one another. The first is that the starting point is a primary degeneration of the nerve fibres, and that the disease of the vessels, the neuroglia and the ganglion cells is secondary; the second view is that the process starts in the vessels, and that an inflammatory process proceeds from these to the neuroglia, the inflammatory products leading to the destruction of the nerve fibres and to changes in the ganglion cells. If it were oftener possible to study very rapidly developing cases of general paralysis—the diagnosis, however, must be absolutely certain—a decision might possibly soon be reached. There is, however, only a small number of such observations. In one case in which there was an early autopsy Greppin found no atrophy of the nerve fibres, while Friedmann, in a case of a little over two months duration, found pronounced changes in the vessels. Mendel is able to report a second such case. These cases, therefore, tell against primary degeneration.

Mendel's assistant, Kronthal, by a special procedure was able to isolate the capillaries in the fresh brain, and in all the cases of general paralysis studied by this method there is shown a great degree of widening of the capillaries, thickening of their walls, and a marked increase of nuclei in these. This speaks still farther for the primary involvement of the vessels. Where all the different elements have been changed by disease, the relation between the atrophy of the nerve fibres and the changes in the vessels is a very varying one; sometimes the changes are relatively proportional, while again one and then another predominates. Under these circumstances it would seem that we could not come to a fixed conclusion with regard to the starting point in men. For this reason Mendel in 1883 undertook experiments to produce in dogs a disease similar to *dementia paralytica* in men. The experiments were repeated with similar results by Lemos, Kusznezow and Fürstner; the last makes the similarity to human paralysis more evident since he finds also in the paralytic dogs disease of the cord and optic nerve atrophy. At the time of Mendel's first experiments the finer methods for the recognition of nerve fibres did not exist, but he has recently gone over the experiments again, directing special attention to how in beginning disease the morbid process first showed itself in the brain; the dogs were therefore killed when they showed the first sure signs of disease. The following are the results of the microscopic examination: Changes in the vessel walls through numerous scattered nuclei, widening of the adventitial spaces, in which numerous nuclei are visible, but *no changes in the medulated fibres and no changes in the ganglion cells.* In dogs, therefore, the disease certainly acts not as a primary degeneration, but as an inflammatory process proceeding from the vessels. To carry a physiological experiment directly over to man, especially in mental diseases, certainly has something misleading, yet we cannot abstain from doing this in doubtful cases. One point from the symptomatology of general paralysis is of importance with regard to this question. In a great number of cases fainting attacks precede the outbreak of the mental disease, and also apoplectiform attacks, conditions which are referred to stagnation in the vascular system. Here then are some changes present at a time when there is absolutely no reason for assuming an atrophy of the fibres. Finally, the conclusion that appears to Mendel justified at this time, even if it cannot be proved with absolute certainty, is that in general paralysis there is first present disease of the vessel walls through hyperæmic stasis in the vessels; exodus of

blood corpuscles and inflammation of the neuroglia follows, leading secondarily to destruction of the nerve elements. General paralysis, then, may be designated as *a diffuse interstitial encephalitis, terminating in brain atrophy.*

Zur pathologischen Anatomie der Dementia paralytica. LUDWIG MEYER. Neurologisches Centralbl., 1890, No. 20, p. 610.

This is a criticism of certain points in the preceding article by Mendel, citing the conclusions that general paralysis is a diffuse interstitial encephalitis, and the conclusions on the microscopic examination of the brain of the dogs. Meyer claims that essentially the same findings have been shown by him to exist in a considerable number of cases, and since Mendel casts doubt on the significance of these observations, either through doubt as to the diagnosis or because of the small number of cases, Meyer reviews his own contributions to the subject of *dementia paralytica* extending over a number of years. In 1858 he claims to have advanced proof that in typical cases the disease takes its course in febrile exacerbations, and may therefore be classed with the chronic febrile diseases as a meningo-encephalitis. The anatomical proof of this was published in the *Centralbl. für med. Wiss.*, 1867, Nos. 8 and 9. The accumulation of the nuclei and cells in the walls of vessels was described, and the change of the ganglion cells in atrophic brains was described as a result of vascular degeneration. Meyer agrees with Mendel that the question of the primary changes can only be settled by a study of those cases with a very rapid development; but this rapidity must without doubt be looked on suspiciously, for since "the diagnosis must be absolutely certain" it is necessary that the time of development as well as of the existence of the typical symptoms should not be too short. Meyer claims that with the limitations just mentioned he has given the essential anatomical changes of the disease in his work on the Pathological Anatomy of Dementia Paralytica (*Virchow's Archiv*, 1873, pp. 270–303.) As far back as then he said "only those changes in the brain can be looked on as pathological which appear and are constantly observed with the first distinct symptoms of disease," and "cases of very short course must serve exclusively as the basis for investigation." "Brains with appearances of atrophy must be excluded, or must be admitted only with great reservation." Meyer found 20 cases answering these conditions; and among other things, the normal brain weights went to indicate that the cases fulfilled the required conditions; and these were further strengthened by some of the phenomena of the course which resembled a severe meningitis, or there were headaches in the beginning, maniacal outbreaks resembling febile delirium, convulsions, paralytic attacks, etc. In all the cases there were early autopsies. There was cell-proliferation of the vessel walls. Meyer's conclusion at that time was that, apart from the chronic meningitis which was not always present, the changes at the beginning of the disease were confined to these vascular changes, which were claimed to be inflammatory. Examination of the substance of the brain gave an entirely negative result.

MANNER OF DEATH.

De la mort dans la paralysie générale. JEAN L. BARAZER. Thèse de Paris, 1890, No. 179.

Barazer considers that the question of the mode of death in general paralysis may be reduced to simpler proportions than in Jamin's thesis of 1887. If it is true that the causes of death may be innumerable, it is also incontestable that the patients always die, or at least almost